Multifocal cutaneous mucormycosis complicating polymicrobial wound infections in a tsunami survivor from Sri Lanka

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A man injured in the tsunami of Dec 26, 2004, returned to Sydney for management of his soft-tissue injuries. Despite broad-spectrum antibiotics, surgical wound debridement, and vigilant wound care, his condition worsened. Muscle and fat necrosis developed in a previously debrided thigh wound, and necrotising lesions arose from previous abrasions. Histological analysis showed mucormycosis in three non-contiguous sites, and *Apophysomyces elegans* was isolated from excised wound tissue. Wound infections, both bacterial and fungal, will undoubtedly add to the morbidity and mortality already recorded in tsunami-affected areas. Other causes of cutaneous mucormycosis might develop in survivors, but this disease can be difficult to diagnose and even harder to treat, particularly in those remaining in affected regions.

The tsunami that struck southeast Asia on Dec 26, 2004, affected 12 countries and has been described as one of the worst natural disasters in modern history. At least 200 000 people died, thousands more were injured, and about 2 million people were displaced.[1]

Basic services such as clean water and sanitation were severely affected in many areas by the tsunami. While health services including hospitals are being re-established, laboratory diagnostic services are scarce or unavailable. Most wound infections in survivors are likely to be caused by bacteria, including water-associated pathogens such as *Aeromonas* spp, which are resistant to commonly used antibiotics. Even if laboratory diagnosis of bacterial or fungal infections were possible, access to antibiotics for less common or multiresistant infections might be limited.

A previously well 56-year-old Australian man was transferred from Sri Lanka to a Sydney hospital for continuing care after he sustained injuries in the tsunami. On Dec 26, 2004, the patient's beach hut collapsed and he was pushed through the debris, ending in a paddy field 1 km away. He repeatedly ingested but did not inhale seawater. His wounds were irrigated with bottled drinking water before being bandaged. Additional first aid and wound debridement were done at a regional hospital before his transfer to Colombo, where his two largest wounds were debrided a further three times. Cefuroxime, metronidazole, gentamicin, and tetanus toxoid were given.

On arrival in Sydney on Dec 31, 2004, the patient was febrile to 38°C but haemodynamically stable. Apart from large areas of deep soft-tissue injury on his right thigh and over his left hip, he had many healing superficial lacerations and abrasions and several superficial limb ulcers with purulent exudate. He was anaemic, with a haemoglobin concentration of 108 g/L (normal range 130–180 g/L), and had a neutrophil level of $14{\cdot}4\times10^9$/L ($1{\cdot}7$–$7{\cdot}0\times10^9$/L). His albumin concentration was 21 g/L (33–48 g/L) but biochemical tests were otherwise normal. Radiographs confirmed no fractures. His chest radiograph and abdominal computed tomography were normal. We empirically administered meropenem, ciprofloxacin and doxycycline to treat bacteria, particularly of soil and water origin. He also received tetanus immunoglobulin and hepatitis A and B immunisation.

The wound on the patient's right thigh measured 25×20 cm and exposed the vastus lateralis muscle and adjacent fascia. These tissues were clean but there was necrotic fat where the wound entered the popliteal fossa. The 20 cm left hip wound exposed the underlying muscle and iliac crest. All wounds were operatively debrided. Initial bacteriological swabs showed polymicrobial growth including: *Aeromonas hydrophila*; a fully sensitive *Pseudomonas aeruginosa*; a multiresistant *Escherichia coli* susceptible only to carbapenems and chloramphenicol; *Achromobacter* sp; and meticillin-resistant *Staphylococcus aureus*. We added vancomycin to his initial antibiotic regimen. On re-exploration of his wounds on day 3 of admission, further fat and muscle necrosis in the right popliteal fossa was debrided.

On day 5 of admission, the patient remained febrile and his neutrophil level rose to $43{\cdot}7\times10^9$/L. Blood cultures were sterile. The sites of left chest wall and right deltoid abrasions developed striking erythema and induration with 5–6 cm of central necrosis (figure 1, A). A histological specimen from the debrided right popliteal tissue showed invasion of necrotic vessel walls by multiple aseptate, broad fungal hyphae (figure 2), associated with extensive arterial thrombosis and necrosis of muscle and fat. These features were consistent with cutaneous mucormycosis. We started liposomal amphotericin B at 5 mg/kg per day. Further necrosis of the distal right semitendinosus and semimembranosus muscles (figure 1, B) needed extensive debridement. Widespread necrotising fasciitis of the left chest wall and right deltoid was resected. This debrided tissue also showed mucormycosis histopathologically.

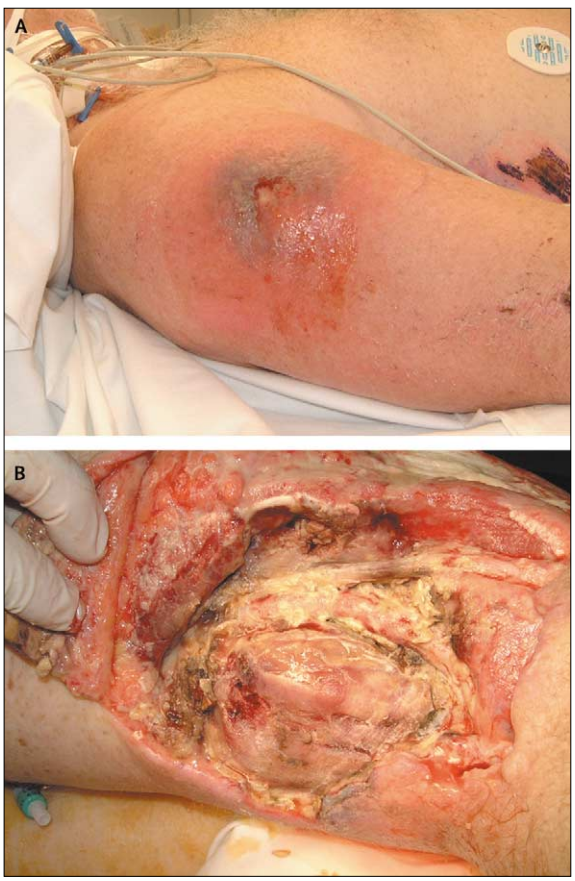

Figure 1: **New right shoulder skin lesion on day 5 of admission before debridement (A), and previously debrided right thigh lesion at day 5 of admission before subsequent debridement (B)**
(A) Lesion arose from previously uninfected abrasion. (B) Right popliteal fossa showing necrotic muscle belly and fat. Patient's thigh is to the left and his calf to the right of the image.

We started adjunctive hyperbaric oxygen therapy. Resected tissue from the right deltoid and left chest wall grew a zygomycete, *Apophysomyces elegans*. This fungus was not isolated from the popliteal tissue, which was debrided after amphotericin B was started. Tissue from other wound sites did not show evidence of mucormycosis on histopathological examination or culture. 3 weeks after his initial injuries, the patient remains hospitalised, with macroscopically clean, healing wounds and no evidence of systemic sepsis.

Most filamentous fungi are free-living (saprophytic) organisms, and they infect people opportunistically after severe immunosuppression, environmental contamination of tissue, or both.[2] Examples of environmental contamination include invasive scedosporiosis after near-drowning[3] and mycotic keratitis after eye injuries.[4] Fungal infections of traumatic wounds can be caused by many organisms, including the zygomycetes that cause mucormycosis. These fungi are ubiquitous in soil and decaying vegetable matter. As well as cutaneous disease,

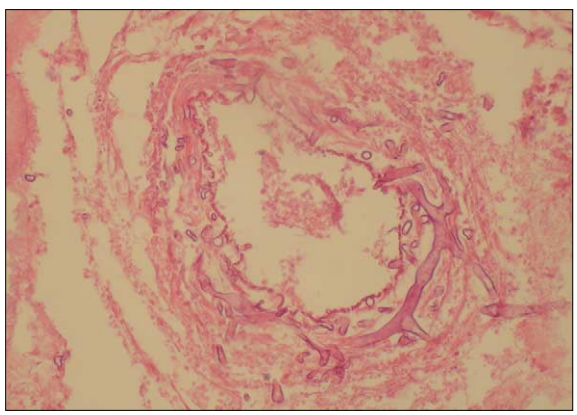

Figure 2: **Debrided popliteal tissue specimen, stained with haematoxylin and eosin**
A broad, ribbon-like, aseptate filamentous fungus is seen, resembling a zygomycete, 5–12 μm in thickness, with vessel wall invasion (original magnification 200×).

mucormycosis can cause rhinocerebral, visceral, and disseminated infections.[2]

Cutaneous mucormycosis is uncommon and can follow either primary inoculation or haematogenous dissemination. Diagnosis on clinical grounds is difficult, and tissue biopsy for histology and culture is usually needed.[5] After a volcanic eruption in Colombia in 1985,[6] 38 patients in Bogotá hospitals developed severe necrotising fasciitis, of whom eight had proven cutaneous mucormycosis. Six of those with this infection died. The best outcomes from cutaneous mucormycosis have been associated with early detection, aggressive surgical debridement, early use of amphotericin B, and correction of predisposing factors.[2,6] Hyperbaric oxygen has been used as an adjunctive treatment,[7] but evidence for its effectiveness is scarce.

Our patient probably acquired mucormycosis from contamination of his wounds at the time of trauma or during first aid measures. Other people injured in the tsunami might also have invasive fungal wound infections, which might go unrecognised unless appropriate tissue specimens are analysed. This case shows that travellers repatriated from affected areas could be good predictors of infections in survivors because they have greater access to advanced diagnostic services than do many of those who remain.

Mucormycosis and other invasive fungal infections should be considered in survivors of natural disasters whose wound infections are progressive despite antibacterial treatment and debridement. Histopathological examination of debrided tissue (including frozen section, if available) is the most rapid way to make an early diagnosis of this disease.

Contributors
A Donaldson and L Choo contributed to patient's care (infectious diseases), literature review, and preparation of the report. D Andresen contributed to microbiology diagnostics and identification of fungus and preparation of the report. A Knox, M Klaassen, and C Ursic contributed to patient's care (surgery) and clinical photography.

L Vonthehoff contributed to anatomical pathology diagnostics and histological photography. S Krilis and P Konecny supervised clinical care of the patient and preparation of the report. All authors contributed to and reviewed the report.

Conflict of interest statement

We declare that we have no conflict of interest.

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